What is claimed is:

1. A mutant thioredoxin molecule, wherein the thioredoxin molecule is resistant to the oxidizing effects of cytokines or reactive oxygen species.

2. The mutant thioredoxin molecule of claim 1, comprising an amino acid alteration that decreases the number of sulfhydryl groups in the molecule as compared to wild type thioredoxin.

3. The mutant thioredoxin molecule of claim 1, wherein the amino acid alteration is a cysteine substitution or deletion.

4. The mutant thioredoxin molecule of claim 2, wherein the substitution or deletion is at residue 32 or an analogous residue.

5. The mutant thioredoin molecule of claim 4, further comprising a substitution or deletion at residue 69 or an analogous residue.

6. The mutant thioredoxin molecule of claim 2, wherein the substitution or deletion is at residue 35 or an analogous residue.

7. A mutant thioredoxin molecule, wherein the thioredoxin molecule is resistant to S-nitrosylation of a SH-group by nitrous oxide.

8. The mutant thioredoxin molecule of claim 7, comprising an amino acid alteration that decreases the number of sulfhydryl groups in the molecule as compared to the wild type thioredoxin.

9. The mutant thioredoxin molecule of claim 8, wherein the amino acid alteration is a substitution or deletion of a cysteine.

10. The mutant thioredoxin molecule of claim 9, wherein the substitution or deletion is at residue 69 or an analogous residue.

11. A method of decreasing, in a target tissue, inflammation induced by a reactive oxygen species, comprising contacting the target tissue with the mutant thioredoxin molecule of claim 1.

12. A method of decreasing, in a target tissue, cytokine-induced inflammation, comprising contacting the target tissue with the mutant thioredoxin molecule of claim 1.

13. A method of decreasing cardiac muscle contractile dysfunction, comprising contacting cardiac myocytes with the mutant thioredoxin molecule of claim 1.

14. The method of claim 13, wherein the cardiac muscle dysfunction is induced by cytokines or reactive oxygen species.

15. A method of decreasing apoptosis in a target tissue, comprising contacting the target tissue with the mutant thioredoxin molecule of claim 1.

16. A method of decreasing insulin resistance in a target tissue, comprising contacting the target tissue with the mutant thioredoxin molecule of claim 1.

17. A method of decreasing cytokine-induced or reactive oxygen species-induced inflammation, insulin resistance, cardiac muscle contractile dysfunction, or apoptosis in a target tissue, comprising contacting the target tissue with a small molecule that blocks a thiol group of a thioredoxin molecule.

18. A method of treating a subject with atherosclerosis or a subject at risk of atherosclerosis, comprising administering to the subject an agent that inactivates endothelial cell dysfunction mediated by a redox-regulated pathway.

19. The method of claim 18 wherein the agent is a mutant thioredoxin molecule, wherein the thioredoxin molecule is resistant to the oxidizing effects of cytokines or reactive oxygen species.

20. The method of claim 18, wherein the redox-regulated pathway is the ASK1 pathway.

21. The method of claim 18, wherein the redox-regulated pathway is the NF-κB pathway.

22. The method of claim 18, wherein the redox-regulated pathway is the p53 pathway.

23. A method of treating a subject with diabetes or a subject at risk for diabetes, comprising administering to the subject an agent that inactivates insulin resistance mediated by a redox-regulated pathway.

24. The method of claim 23, wherein the diabetes is Type 2 diabetes.

25. The method of claim 23, wherein the agent is a mutant thioredoxin molecule, wherein the thioredoxin molecule is resistant to the oxidizing effects of cytokines or reactive oxygen species.

26. The method of claim 23, wherein the redox-regulated pathway is the ASK1 pathway.

27. The method of claim 23, wherein the redox-regulated pathway is the NF-κB pathway.

28. The method of claim 23, wherein the redox-regulated pathway is the p53 pathway.

29. A method of treating a subject with an apoptotic disease or at risk for an apoptotic disease, comprising administering to the subject an agent that inactivates apoptosis mediated by a redox-regulated pathway.

30. The method of claim 29, wherein the apoptotic disease is a neurodegenerative disease.

31. The method of claim 29, wherein the agent is a mutant thioredoxin molecule, wherein the thioredoxin molecule is resistant to the oxidizing effects of cytokines or reactive oxygen species.

32. The method of claim 29, wherein the redox-regulated pathway is the ASK1 pathway.

33. The method of claim 29, wherein the redox-regulated pathway is the NF-κB pathway.

34. The method of claim 29, wherein the redox-regulated pathway is the p53 pathway.

35. A method of treating a subject with cardiac dysfunction or a subject at risk of cardiac dysfunction, comprising administering to the subject an agent that inactivates cardiac dysfunction mediated by a redox-regulated pathway.

36. The method of claim 35, wherein the cardiac dysfunction is selected from the group consisting of myocardial infarction, cardiomyopathy, arterial hypertension, and heart failure.

37. The method of claim 35, wherein the agent is a mutant thioredoxin molecule, wherein the thioredoxin molecule is resistant to the oxidizing effects of cytokines or reactive oxygen species.

38. The method of claim 35, wherein the redox-regulated pathway is the ASK1 pathway.

39. The method of claim 35, wherein the redox-regulated pathway is the NF-κB pathway.

40. The method of claim 35, wherein the redox-regulated pathway is the p53 pathway.

41. A method of treating a subject with an angiogenesis-dependent disease or a subject at risk for an angiogenesis dependent disease, comprising administering to the subject an agent that blocks thioredoxin-inactivation of apoptosis, wherein the apoptosis is mediated by a redox-regulated pathway.

42. The method of claim 41, wherein the angiogenesis dependent disease is an inflammatory disease.

43. The method of claim 42, wherein the inflammatory disease is rheumatoid arthritis.

44. The method of claim 41, wherein the angiogenesis dependent disease is cancer.

45. The method of claim 41, wherein the agent oxidizes thioredoxin.

46. The method of claim 41, wherein the redox-regulated pathway is the ASK1 pathway.

47. The method of claim 41, wherein the redox-regulated pathway is the NF-κB pathway.

48. The method of claim 41, wherein the redox-regulated pathway is the p53 pathway.

49. A method of diagnosing an angiogenesis dependent disease in a subject or of identifying a subject at risk for developing the angiogenesis dependent disease, comprising detecting, in a biological sample of the subject, levels of reduced thioredoxin, wherein the angiogenesis dependent disease is indicated by an elevated level of reduced thioredoxin as compared to control levels.

50. The method of claim 49, wherein the angiogenesis dependent disease is cancer.

51. The method of claim 49, wherein the angiogenesis dependent disease is arthritis.

52. A method of diagnosing an apoptotic disease in a subject or of identifying a subject at risk for developing the apoptotic disease, comprising detecting, in a biological sample of the subject, levels of oxidized thioredoxin, wherein the apoptotic disease is indicated by an elevated level of oxidized thioredoxin as compared to control levels.

53. The method of claim 52, wherein the apoptotic disease is a neurodegenerative disease.

54. A method of screening a subject for a genetic risk of an angiogenesis dependent disease, comprising detecting a gene that encodes a mutant thioredoxin molecule.

55. The method of claim 54, wherein the mutant thioredoxin molecule is resistant to the oxidizing effects of cytokines or reactive oxygen species.

56. The method of claim 55, wherein the mutant thioredoxin molecule comprises an amino acid alteration that decreases the number of sulfhydryl groups in the molecule as compared to wild type thioredoxin.

57. The method of claim 56, wherein the amino acid alteration is a cysteine to serine substitution.

58. The method of claim 57, wherein the substitution is at residue 32.

59. The method of claim 57, wherein the substitution is at residue 35.

60. A method of screening a subject for a genetic risk of an apoptotic disease, comprising detecting a gene that encodes a mutant thioredoxin molecule.

61. The method of claim 60, wherein the mutant thioredoxin molecule is resistant to S-nitrosylation of a SH-group by nitrous oxide.

62. The method of claim 61, wherein the mutant thioredoxin molecule comprises an amino acid alteration that decreases the number of sulfhydryl groups in the molecule as compared to wild type thioredoxin.

63. The method of claim 63, wherein the amino acid alteration is a substitution or deletion of a cysteine.

64. The method of claim 63, wherein the substitution or deletion is at residue 69.